Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services

Emergency Programs

Number 13-2 June 1985

In This Issue

Current Events

VVND in Florida and Puerto Rico

Exotic pet birds were found to be infected with velogenic viscerotropic Newcastle disease (VVND) on three Florida premises during the period from January 17 to February 26, 1985. Two of the positive premises were commercial wholesale facilities; the third was a private aviary.

Tracing of sales from one of the positive premises disclosed an additional case in Puerto Rico. The tracing of birds from the Puerto Rico premises and from the wholesale dealers in Florida to 25 additional States failed to detect additional cases.

It appears that "yellow-nape season" is here again. A yellow-naped Amazon parrot recently submitted to a diagnostic laboratory by a veterinarian in California was positive for VVND. The dead bird was found on his doorstep and cannot be traced. (Dr. K. A. Hand, 301 436-8065).

Screwworm Program Update

On January 31, 1985, the Mexico-U.S. Screwworm Eradication Program officially ended its eradication efforts and began establishing a defensive barrier at the Isthmus of Tehuantepec. The sterile fly barrier is designed to prevent the return of this insect to the areas of Mexico and the United States previously freed by the eradication program. (See June 1983 issue, 11-2)

The changes required to redirect the objective of the program were primarily in three functions: relocation of field inspectors, reduction of the production of sterile flies, and intensification of the control of animal transport through the barrier zone. A total of 107 inspectors from the four other regions were transferred to the barrier region to intensify the field inspection, both in the barrier and in areas immediately adjacent to it. The production of sterile flies was limited to the number required to maintain the barrier zone.

Inspectors at quarantine stations have inspected and either dipped or sprayed 62,281 animals since January 1, 1985. Of these animals, 4,523 were found to have wounds and 10 animals had screwworms. All wounds were treated and the animals with screwworms were quarantined until they could be safely transported.

A total of 12 cases of screwworm myiasis have been reported since the barrier zone program was initiated. There have been two cases north and west of the barrier and 45 cases south and east of the barrier. (Dr. Robert Reichard, Co-Director, Aftosa Program, U.S. Embassy, Panama, Box E, APO Miami, Florida 34002).

Avian Influenza Surveillance Completed

The avian influenza area quarantines in Virginia and Pennsylvania were lifted September 14 and October 4, 1984, respectively. (See 13-1). A 6-month surveillance program was deemed necessary for the ensuing fall and winter months to detect any subtype H_5N_2 influenza virus activity.

The surveillance periods ended in Virginia on March 15 and in Pennsylvania on April 2, 1985.

In Virginia, surveillance activities did not detect any H_5N_2 virus or antibodies. However, avian influenza antibodies for subtype H_1N_1 were found in slaughter turkeys and H_7N_3 in a backyard flock. Also, subtype H_2N_3 virus was isolated from one turkey flock, and subtype $H_{10}N_8$ virus was isolated for the second time from another. The latter flock experienced subtype $H_{10}N_8$ virus in April 1984 and all turkeys on the premises were depopulated. Only ducks were allowed to remain. After the second occurrence, all fowl on the premises were depopulated by the owner.

Surveillance activities in Pennsylvania detected two seropositive backyard flocks, but no avian influenza virus.

Surveillance was accomplished by every-other-week egg collections from layer flocks; weekly dead bird collections from turkey, broiler, and pullet flocks; and blood and swab sampling at slaughter from poultry at slaughter plants in the formerly quarantined areas. In addition, spent hens in Pennsylvania were bled within 2 weeks before slaughter, to precede the usual practice of slaughtering out of State, especially in New Jersey. Quantities per sampling were a dozen eggs for a small flock, 30 eggs for a large layer house, and a maximum of 30 dead birds or 30 blood and swab samples. Egg yolk and serum tests were conducted in the State diagnostic laboratories using the agar-gel immunodiffusion (AGID) test. Specimens for virus isolation attempts were sent to the National Veterinary Services Laboratories (NVSL), Ames, Iowa.

Suspicious laboratory results on surveillance specimens triggered an immediate investigation of the source flock. All specimens collected in the investigation, plus those found to be suspicious at the State laboratories, were sent to NVSL for confirmatory examination. (Dr. Arthur E. Hall, 301 436-8073).

Maryland Avian Influenza

In December 1984, poultry were seized from a poultry dealer in Washington D.C., who had been detained for violating local sales licensing laws. The chickens were sick and some were dying. Avian influenza virus subtype H_5N_2 was isolated from these chickens at the State laboratory in Salisbury, Maryland. Other poultry species from the seized group were given by the local humane society to a couple of backyard flock owners. The virus was isolated from chickens in one of these backyard flocks. As a result, the dealer's flock and two flocks that had received birds from the dealer were depopulated. Tracings from the dealer's flock led to the investigation of 250 backyard flocks in Virginia, Pennsylvania, New Jersey, Delaware, and Maryland. No evidence of H_5N_2 was detected in these flocks. Of the traced flocks, 121 were in Pennsylvania, primarily in the formerly quarantined area. Failure to find the virus or antibodies in these backyard flocks gave added assurance that the disease had been eradicated.

The subtype H_5N_2 virus isolated in Maryland was found to be nonpathogenic for poultry at NVSL. Examination of the virus structure at the World Health Organization Influenza Center, St. Jude Children's Research Hospital, Memphis, Tennessee, indicated a similarity to the April 1983 isolates from Pennsylvania. (Dr. Arthur E. Hall, 436-8073).

Role of NADDS in Emergency Disease Detection

The National Animal Disease Detection System (NADDS) began 2 years ago as a pilot activity for the Animal and Plant Health Inspection Service (APHIS). Federal and State veterinarians in five States are now collecting data on diseases, economic losses, and other health-related events occurring on randomly selected farmsteads.

After the methods are refined and established, NADDS will systematically expand into a national system in which inferences drawn from collected data can be applied to entire industries at risk. Because of the sampling technique used and knowledge of animals at risk, relatively few premises in each State need to be sampled; yet, in the aggregate, results will accurately reflect the disease picture for an entire population. The information derived from a national surveillance system will serve as the basis to make accurate estimates on the prevalence, incidence, trends, and economic impact of livestock and poultry diseases in the United States.

NADDS is an example of an active surveillance system designed to capture reports of all disease conditions of livestock and poultry. In NADDS we stimulate detection and reporting through randomly selected farms, ranches, and flocks. On farm data are collected monthly through producer interviews by specially trained regulatory veterinarians. These data are recorded on standardized reporting forms by use of a standardized nomenclature to describe the problems. Both numerators (herds and flocks experiencing diseases and conditions) and denominators (population at risk stratified by size and production type) are measured and used to calculate the occurrences of health-related events in the populations at risk.

Active surveillance is concerned with the collection and tabulation of occurrences of diseases and conditions, the analysis and interpretation of data, and the generation of timely, statistically valid reports for use by people who need the information. In contrast, passive surveillance is usually concerned with only a particular portion of the numerator, and there is little knowledge of the denominator. Examples of passive surveillance systems are the reporting systems used in diagnostic laboratories, slaughter plants, veterinary hospitals, and research institutions. Both active and passive systems are needed in a total surveillance effort. Although these systems have different objectives and monitor different populations, both are compatible and essential.

By raising a general awareness among producers and veterinarians to the nature of active and passive surveillance and the need to work together in a cooperative effort to protect American agriculture, NADDS nationwide will enhance emergency disease detection. Livestock and poultry producers who are involved in NADDS are responsible for recording much of the disease and economic data, and therefore have a much better appreciation and awareness of disease problems in their herds and flocks. Then, with regular publication of results of NADDS in farm journals, this awareness is extended to a large number of producers and will help in the recognition and monitoring of all diseases, including foreign animal diseases (FAD).

Our principal line of defense in detecting an FAD incursion resides in passive surveillance systems. Laboratory diagnosticians and private practitioners will probably be the first group to come into contact with an FAD. The probability that NADDS will detect the first case of an FAD in the United States is extremely remote. NADDS is not designed, nor is it intended, to find such rare occurrences. We estimate that the national system will need only 5,000 to 6,000 herds and flocks across the United States to include all strata of beef cattle, dairy cattle, hogs, sheep, and poultry. These participants represent only a small fraction of the 1.7 million U.S. farmsteads that contain livestock or poultry.

NADDS will also expedite and strengthen emergency animal disease eradication operations. If an FAD enters the United States and becomes established, then an active surveillance system can function to define the movement and extent of spread of such an outbreak. With veterinarians trained and participating in onfarm data collection and with a knowledge of populations at risk, an infrastructure exists in which veterinarians in emergency disease control and eradication operations can increase sampling and surveillance in strategic areas and take on an FAD detection function.

At the same time, NADDS has a laboratory-based validating component, with the necessary linkage to diagnostic facilities to screen for foreign animal diseases in case of an emergency outbreak situation. In the past, during an FAD outbreak, nonaffected areas or regions of the country often had an unstructured and nonstandardized protocol for detection and

surveillance of such diseases. This increased their vulnerability to the spread of foreign animal diseases to their areas without their knowledge and risked delayed response time in reacting to an emergency.

Active and passive systems can play important roles in the surveillance of both domestic and exotic diseases. With the implementation of NADDS, we will, for the first time, be able to reliably define the economically significant disease problems that are reducing production efficiency. We believe that our livestock and poultry are among the healthiest in the world. However, accurate information from NADDS on the disease problems of the relatively small percentage of unhealthy animals will serve as the basis to make better and more cost-effective decisions on herd health. Together, information from the diagnostic laboratories, veterinary practices, and NADDS will provide a complementary network to serve in a total surveillance effort to monitor our gains and protect animal health. (Dr. L. J. King, 301 436-8087)

Cured Pork Imported Under New Rule

Minimum protein levels were established in April 1985 for both imported and domestically produced ham and other cured pork products. The protein level is figured on a protein fat-free basis. This means that the protein content is based on the protein level of the lean portion of the product, not the entire product. The method formerly used was less accurate and consisted of measuring the amount of curing solution used in processing. Compliance with the Federal standards assures wholesome, accurately labeled products.

Residue testing of imported meat and poultry products is also equivalent to the testing required for the domestically produced products, under provisions of the 1981 Farm Bill. In 1983, the Food Safety and Inspection Service (FSIS) conducted special reviews of major exporting countries. Results of this review justified declaring six countries that did not satisfy the Farm Bill requirements ineligible to export meat. Four of the six were reapproved after the deficiencies had been corrected.

USDA requires that imported meat and poultry be processed under inspection programs equal to the U.S. program. Imported products are reinspected at ports of entry to further ensure compliance with USDA requirements. (Sharin Sachs, FSIS, USDA, Information and Legislative Affairs Information Branch, Washington, D.C. 20250; 202 447-9113)

Effect of Drying on FMD Virus Survival

Foot-and-mouth disease (FMD) virus survived for 2 years in vesicular fluid dried slowly at 18°C and kept dry at the same temperature. However, if the temperature or humidity were increased, or the suspending medium was changed, the virus survived for only a few days or weeks. FMD virus also survived for several months in cattle fodder, bran, sugar, or hay stalks, when dried naturally in a cool, dry environment protected from light. This lengthy survival was thought to be due to the dry, chemically inactive environment provided by the material surrounding the virus particles. Survival time was shortened when FMD virus was dried on surfaces of glass, hair, wool, or sand. (Summary of literature in the Emergency Programs

Information Center Data Bank, Veterinary Services, APHIS, USDA, by Dr. Michael Gilsdorf, 301 436-8379).

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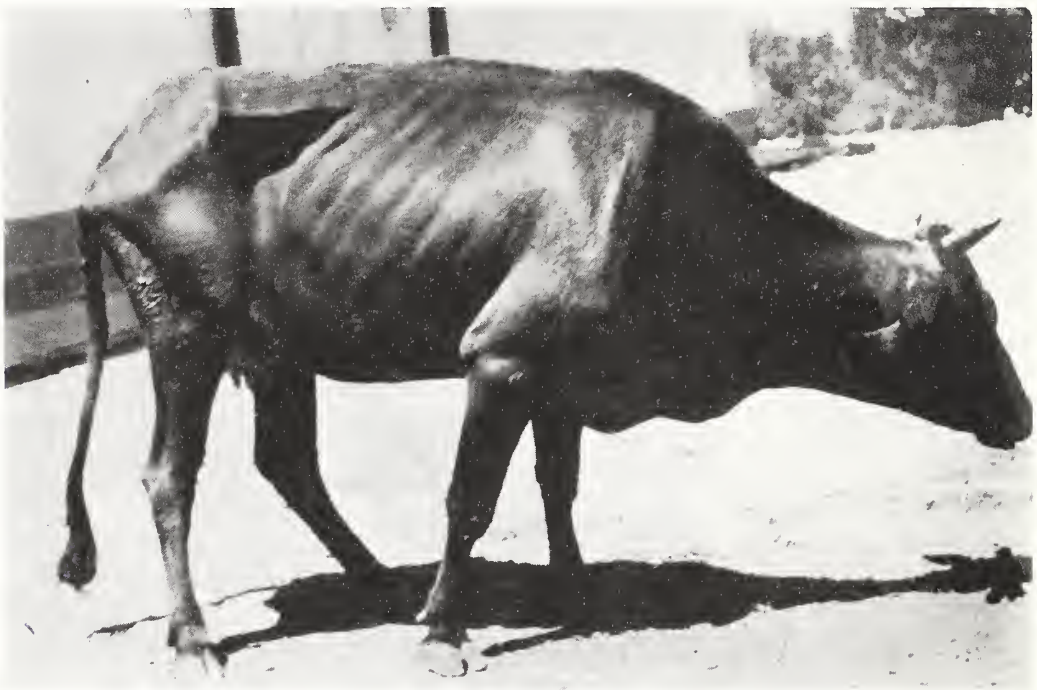
Asiatic Hemorrhagic Septicemia

Hemorrhagic septicemia (HS), has emerged as one of the most important animal diseases affecting cattle and buffaloes throughout Asia. The disease is of considerable economic importance as it affects both draft power and food sources.



In order to understand HS, it is important to first understand that it is caused by heterogeneous bacterial species, Pasteurella multocida, displaying considerable diversity in host predilection, pathogenicity, biochemical activity, colony morphology, and antigenicity.

HS Serology

R. S. Roberts (1947) identified four immunotypes by passive protection tests in mice. He designated them as types I, II, III, and IV. His type I strains were all recovered from animals with HS.

G. R. Carter (1955) identified four different capsular antigens of P. multocida by an indirect or passive hemagglutination procedure in which the capsular substances were adsorbed onto red cells. These capsular types are designated A, B, D, and E. Of these, only B and E have been shown to cause HS.

S. Namioka and M. Murata (1961) employed acid-heated cells and agglutination absorption to show different O or somatic antigens. They identified serotypes by a number representing the somatic antigen, followed by a letter representing the Carter type. The serotype that causes HS in Southeast Asia was designated 6:B. In all, 11 somatic varieties have been identified.

K. L. Heddleston and associates (1972) found they could identify different serological varieties with a gel diffusion precipitin test. Their 16 somatic types were identified by anti-pasteurella sera that were prepared in chickens. This system of designating serotypes was an extension of the earlier classification of P. A. Little and B. M. Lyon (1943). Their type 2 could be either 6:B or 6:E, as the capsular antigen was not recognized. The failure to include the capsular type and the considerable cross-reactions encountered have limited the application and usefulness of this system. However it has been of value in the serological analysis of fowl cholera strains, all of which belong to capsular type A.

At least six different systems have been used to classify P. multocida. (See table).

Systems Adopted for Typing Pasteurella multocida and the Position of Hemorrhagic Septicemia Serotypes

Authors	Typing Technique	Type Designation	Position of HS Types
Little & Lyon (1943)	Slide agglutination and passive mouse protection test	1, 2 & 3	2
Roberts (1947)	Passive mouse protection test	I-IV	I
Carter (1955)	Capsular typing by indirect hemagglutination test using heat labile (56^{o}C-30 min) antigen	A, B, D & E	B & E
Namioka & Murata (1961)	Simplified capsular typing by slide agglutination test using fresh cultures	A, B, D & E	B & E
Namioka & Murata (1961)	"Somatic" typing by agglutination test using HCl treated cells	1-11	6
Heddleston et al. (1972)	Gel-diffusion test using heat stable (100^{o}C for 1 hour-supernate) antigen	1-16	2 & 5

The use of a "somatic-capsular" typing scheme (Namioka-Carter) was recommended at the Food and Agriculture Organization (FAO-APHCA) workshop on HS held in Colombo, Sri Lanka in 1979. The prevalent serotype in Africa is 6:E. Two countries, Egypt and Sudan, have reported both 6:B an 6:E. Type 6:B was isolated from bison in the United States. Although HS antibodies have

been reported in Mexico, Central America, and South America, there are no reports of either serotype being recovered. It is possible that these countries may be reporting other forms of bovine pasteurellosis and not specifically HS.

Asiatic hemorrhagic septicemia is normally associated with wet, humid weather. In most cases, the incidence increases with the onset of the rainy season (monsoon). Wet areas (buffalo populated) are the most affected, and dry areas (cattle-populated) are usually less affected. Incidence is highest in herded buffaloes, while buffaloes dispersed in small numbers on small farms are usually only lightly affected. Buffaloes appear to be more susceptible than cattle. Young buffaloes, 6 months to 2 years of age, are most susceptible, especially in enzootic areas. Repeated vaccination may account for the resistance of older animals, which are quite susceptible if unvaccinated. HS occurs mostly in areas where animal husbandry practices are primitive, and where veterinary services and disease reporting procedures are poorly developed. Reported losses are probably lower than actual losses throughout the Asian area.

HS Hosts

Asiatic HS occurs principally in buffaloes and cattle under natural conditions. However, several sporadic outbreaks of acute septicemic pasteurellosis in pigs associated with HS serotype 6:B, have been reported in Sri Lanka. One of these was associated with the feeding of bovine blood from an abattoir to pigs. This isolate produced typical HS in cattle. Similar reports of sporadic incidence in pigs caused by organisms serotyped as 6:B have come from Thailand and Malaysia. India has also reported cases of type B infection in swine, but without somatic antigen determination.

Septicemic pasteurellosis in sheep and goats has been reported to be of Roberts type I. However, type I is not necessarily the same as 6:B. An Australian non-HS strain (Strain 989) is type B for capsular antigen, but the somatic antigen is Namioka's 11 instead of 6. India and Iraq have identified a few strains of *P. multocida* from sheep as type B, but the somatic antigen is not yet known.

Septicemic pasteurellosis, caused by serotype 6:B, has been recorded in elephants in Sri Lanka in conjunction with outbreaks of HS in cattle and buffaloes.

Sporadic outbreaks of septicemic disease associated with *P. multocida* and typed as Roberts' type I, Carter's type B, or serotype 6:B have been recorded in species other than cattle and buffaloes. The disease associated with these types is referred to as "septicemic pasteurellosis." The term, "hemorrhagic septicemia" is usually reserved for the disease in buffaloes and cattle.

HS Pathogenesis and HS Pathology

As hemorrhagic septicemia occurs mostly in regions where husbandry practices are primitive and animals are reared under semiwild conditions, often the only sign reported is sudden death. The course of the disease is usually from 1 to 3 days.

Signs most frequently observed are dullness, fever up to 41.7°C, salivation, nasal discharge, and painful edematous swelling beginning in the submandibular area and spreading to the brisket and perineum, followed by progressive respiratory distress. The affected animal then becomes recumbent and usually dies. Gastroenteritis has been noted in calves. Recovery is rare, especially in buffaloes.

Buffaloes with HS usually die quicker than cattle and show fewer lesions. A few atypical syndromes have been recorded; for example, a syndrome associated with edema and lameness of the forelimbs has been reported in Burma. In Sri Lanka, a pneumonic pasteurellosis and lameness, caused by serotype 6:B, has been recorded in young buffaloes in enzootic areas during the HS season.

The extent of the gross pathological lesions depends on the course of the disease. Animals which died within 24 to 36 hours after experimental infection had generalized congestion of the lungs and widespread petechial hemorrhages, most pronounced at the base of the heart, in the abomasal wall, and, to a lesser extent, at the serosal surface of the intestines. When the animals died within 48 hours, hemorrhages were more severe and fibrinous pericarditis was present. After 72 hours, there was extensive consolidation of the lungs, with lobulation due to marked thickening of the interlobular septa, sero-fibrinous pericarditis, pleurisy, and adhesions between the pericardium and pleura.

HS Epidemiology

In affected countries, there are areas of high, moderate, and low incidence, particularly associated with water courses, river deltas, and dry areas. Few Asian countries have produced evidence to support the widely held idea that losses due to HS are higher in buffaloes than cattle. A survey in Sri Lanka indicated that the herd infection rate was much the same in cattle and buffaloes, but the morbidity rate within affected herds was considerably higher in buffaloes. A notable exception to this is in the Bali breed of cattle (Bos banteng), which is reported to be highly susceptible. Little information is available concerning European and crossbred cattle, whose numbers are increasing in Asia. As these animals are highly valued, they are usually vaccinated against HS.

In areas where regular seasonal outbreaks occur, mortality in individual outbreaks is low and confined to young animals. When occasional sporadic outbreaks occur outside the enzootic areas, mortality is high and animals of all ages are affected. This relationship appears to have an immunological basis, since a high percentage of animals in enzootic areas have naturally acquired immunity. The chief source of naturally acquired immunity in enzootic countries is subclinical or "arrested infection." In situations where no vaccination is practiced, the morbidity and mortality patterns are largely governed by the proportion of naturally immune to nonimmune animals at the time of the outbreak. It is estimated that 10 percent of cattle and buffalo in Southeast Asia have naturally acquired immunity. In a survey of 925 animals in Sri Lanka, the incidence of

naturally acquired immunity in areas of low, moderate, and high HS incidence was 0.47, 7.2, and 36.1 percent, respectively.

HS Carriers

Some healthy cattle and buffaloes carry pathogenic pasteurella in their nasopharynges. A correlation has been established between the incidence of HS and the carrier rate. During an epizootic of HS in an Indian village, 7.5 percent of healthy animals were carriers, but none were detected in the same village 40 days later. The proportion of carriers seems to vary with the incidence of HS, ranging from no carriers in disease-free areas to 6 to 8 percent of the total cattle and buffaloes in high incidence areas. This has been corroborated in studies in India and Sri Lanka. It has also been found that the carrier rate is highest in clinically healthy animals immediately after an epizootic and it declines rapidly in 4 to 9 weeks. Regular vaccination may eliminate the carrier status.

An eradication program with intensive annual vaccination has been launched on the Indonesian Island of Lombok. After the first year there were reported to be 2.3 percent carriers. No carriers were detected after the second year. Studies in Sri Lanka have indicated that the carrier state is transient and different animals are carriers at different times in the first few weeks following an outbreak. It has also been found that some animals free of virulent pasteurella in the nasopharynx harbor the organisim in their retropharyngeal lymph nodes. It is not known how long the organisms persist in the retro-pharyngeal lymph nodes or whether persistence is related to immunological deficiency.

HS is spread from carriers by both direct and indirect contact. The causal virus reportedly does not survive more than 2 to 3 weeks in the soil or on pastures.

HS Laboratory Diagnosis

Specimens for the laboratory diagnosis of HS should include smears of edema fluid and blood, edema fluid, lymph nodes, whole blood, blood collected in ethylenediaminetetracetic acid (EDTA) for animal inoculation, and a section of long bone or marrow. All samples except smears should be kept refrigerated until examined.

In the developing countries in southeastern Asia, most laboratory confirmation is based on cultural characteristics, bacterial cell morphology, staining characteristics, and the results of animal inoculation tests.

Mice and rabbits are both highly susceptible to HS types 6:B and 6:E. Typing of isolates is seldom done because most countries lack the necessary reference strains and sera. Serotypes 6:B and 6:E may be identified with immune rabbit sera in the mouse protective test. Somatic antigen identification seems feasible in only one or two Asian countries.

HS Therapy

Treatment of HS is rarely attempted because the success rate is known to be very poor after typical signs appear. Treatment is effective only very early in the course of HS. Rectal temperatures should be checked twice daily in all animals that

have been in contact with HS-infected animals after the first case has been reported. Antibiotic therapy should begin immediately if a temperature rise is noted. There are few reports of antibiotic resistance in HS strains of P. multocida. Strains from Malaysia, Indonesia, Thailand, Burma, India, and Sri Landa, when tested in Sri Lanka, were sensitive to 10 common antibiotics, except that the Thai strain showed partial resistance to streptomycin. Treatment with hyperimmune serum after the onset of infection reportedly has no effect. Hyperimmune serum given before experimental challenge or natural exposure may protect cattle for up to 1 month.

HS Control

In all countries where HS occurs, vaccination is accepted as the method of control. Most vaccines for this purpose are produced in the countries in which they are used. Most countries use broth bacterin or alum-precipitated vaccine (APV), while a few use oil adjuvant vaccine (OAV). Thailand alone uses an aluminum hydroxide gel vaccine. Most of the vaccines are not standardized products. Vaccines produced in different countries differ in the strain of organism used, the method of cultivation, and the bacterial content per dose. India uses a selected vaccine strain (P52) which is believed to possess special immunogenic properties. Indonesian countries use indigenous strains. Five strains from five different regions of the country are used in Malaysia.

The mechanism of immunity to HS is apparently antibody-mediated. Cell-mediated immunity to HS has not been demonstrated. The immune status of an animal depends on the amount of antibody in the blood and tissue fluids. When HS antibodies drop to a very low level, the animal again becomes susceptible. (Dr. James T. Cavanaugh, Veterinary Attache, U.S. Embassy, Manila, The Philippines)

World Animal Disease Roundup

An overview of the world animal disease situation shows that **African swine fever** (ASF) is receiving the most attention at this time. It invaded Belgium by the most classical method: food scraps brought from an affected country (Spain) by a tourist. With the completion of depopulation on 9 infected and 18 exposed premises, there is hope that the problem has been contained. Whether the same can be stated for a recent outbreak of **foot-and-mouth disease** (FMD) in Italy appears doubtful. By March 27, 1985, a total of 107 cases had been counted in that country since the first case appeared in November of 1984. No FMD has been reported from Africa for some time, but this may be the result of a lack of reporting and not an indication that the disease has disappeared. In South America, FMD continues to be reported in all the places where it has been more or less endemic. However, about a year has passed since FMD was last seen in Chile.

Rinderpest was reported from Togo and Mali. Contrary to our report in the March 1985 issue of this publication (13-1:6) that an African campaign against rinderpest was to start on December 5, 1984, the campaign has been postponed. The process of obtaining required funding and making administrative and logistical preparation is slower than anticipated. Veterinary

Services has given advisory assistance to the Government of Somalia on a rinderpest-related issue. Somalia has difficulty in obtaining approval to export slaughter cattle to other countries because of the presence of rinderpest in Africa. The Animal and Plant Health Inspection Service is involved with plans for the development of appropriate facilities and handling methods which would enable Somalia to guarantee that their exports are free of rinderpest virus.

No significant change in the number of cases of **hog cholera** was reported from major swine producing areas. No reports were received on **swine vesicular disease...**(Dr. Hans J. Seyffert, 301 436-8285).

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Focus on: Bovine Theileriosis

Theilerial parasites occur in many ruminant species, including domestic and wild Bovidae. These tick-borne protozoa multiply in cattle by schizogony in lymphocytes, followed by quadruple division of the piroplasms in red cells, to form typical "maltese crosses." Transmission by ticks is transstadial (across stages of the tick growth cycle), the infection being either acquired by the larva and transmitted by the nymph, or acquired by the nymph and transmitted by the adult. These features, and certain aspects of the parasitic life cycle in ticks, differentiate Theileria from the related genus Babesia.

Bovine theilerial infections occur on all continents and are caused by at least six different species, varying from highly virulent pathogens to organisms causing asymptomatic infection, as summarized in the following table.

Bovine theileriosis clearly is not a single disease. Theilerial species are most reliably differentiated by serological methods. Morphological characteristics of piroplasms and schizonts may also be of help, but are inconclusive except for the piroplasms of T. orientalis and T. velifera. The tick vector involved is also a useful indicator of parasite species.

Species and Distribution

The African wild buffalo (Syncerus caffer) is almost certainly the original host of at least three theilerial species: T. parva, T. mutans, and T. velifera. Another African ruminant, the eland antelope (Taurotragus oryx), appears to be the original host of T. taurotragi. Likely candidates for the original hosts of T. annulata and T. orientalis are the ancestors of domestic cattle and/or of domestic Asian buffalo.

Theileria parva occurs as biologically different types. The original parasite of buffalo, called T. parva lawrencei, causes Corridor disease in cattle, with few lymphocytic schizonts and hardly any piroplasms, so that the disease is normally self-limiting in cattle.

Bovine Theilerial Species

Species	Main known hosts	Main known tick vectors	Disease caused	Distribution
T. annulata	Cattle, Asian buffalo	Hyalomma spp.	Mediterranean or tropical theileriosis	Asia, Europe, Africa
T. mutans	Cattle, African buffalo	Amblyomma spp.	Benign African bont tick theileriosis	Africa, Caribbean
T. orientalis	Cattle, Asian buffalo	Haemaphysalis spp.	Oriental theileriosis, benign cosmopolitan theileriosis	Asia, Australia, Europe, Africa, America
T. parva	Cattle, African buffalo	Rhipicephalus spp.	East Coast fever, Corridor disease, Zimbabwean malignant theileriosis	Africa
T. taurotragi	Cattle, eland antelope	Rhipicephalus spp.	Benign African rhipicephaline theileriosis	Africa
T. velifera	Cattle, African buffalo	Amblyomma spp.	Non-pathogenic	Africa, Caribbean

Classical East Coast fever (ECF) is caused by strains of T. parva parva that have been transformed by passages in cattle. There are large numbers of schizonts and piroplasms in acute cases.

Parasite numbers in so-called Zimbabwean malignant theileriosis, T. parva bovis infection, are intermediate between those seen in Corridor disease and ECF.

T. parva is found from the southern Sudan down to South Africa, and from the Indian Ocean littoral--where the name East Coast fever originated--as far west as parts of Zaire. It probably occurred wherever the wild buffalo and the main vectors of T. parva, Rhipicephalus appendiculatus and related tick species such as R. zambeziensis, coexisted. The transformed parasites, T. p. parva and T. parva bovis, have also spread to regions without buffalo. The potential distribution area of classical ECF, the most devastating form, includes all regions where the vector ticks occur, including large parts of South Africa, Zimbabwe, Mozambique, and the island of Mauritius.

East Coast fever has been eradicated from most of southern Africa. The agent of Corridor disease persists even in South

Africa in wild buffalo, while Zimbabwean malignant theileriosis is also common in parts of Zimbabwe. Strains of T. parva bovis have been isolated as far to the north as Rwanda and are certainly not limited to Zimbabwe.

Mediterranean or tropical theileriosis is neither limited to the Mediterranean area nor to the tropics. T. annulata is found in North Africa from Morocco to Egypt, where its distribution reaches down along the Nile to the central Sudan; in southern Europe, with the greatest impact on the Balkan peninsula and southern Russia; in Asia in parts of the southern USSR, the Near East and Middle East, the Indian peninsula, and possibly part of western China. The main vectors are Hyalomma detritum and H. anatolicum anatolicum, but several other species of the genus are actual or experimental vectors.

Theileria orientalis, formerly T. sergenti, occurs in southern Asia, Iran, India, Malaysia, Vietnam, Indonesia, eastern Asia as far north as the littoral of eastern Siberia, Korea, Japan, eastern Australia, and New Zealand. In Europe, the species occurs as far north as England and northern Germany. It may be in parts of northern Africa, while definite identifications have been made in Africa, Ethiopia, and Burundi. The species has been identified in the United States only in Texas. Certain reports on bovine theilerias in Latin America may also refer to T. orientalis. Known vectors all belong to the genus Haemaphysalis: H. longicornis in Asia and H. punctata in Europe are the main vector species, but by no means the only ones. The vectors in America and tropical Africa are unknown, but are unlikely to be Haemaphysalis ticks.

Theileria mutans (long a catchall name for benign theilerias throughout the world) is probably limited to subsahara Africa, islands near Africa (such as Madagascar and Mauritius) and the Caribbean area where it has been diagnosed on the French island of Guadeloupe. Its vectors include at least six African Amblyomma species, one of which (A. variegatum) was introduced with T. mutans, into the Caribbean basin with African cattle in the 19th century.

Theileria taurotragi probably occurs wherever its known vectors, Rhipicephalus appendiculatus and R. pulchellus, coexist with their natural host, the eland antelope. Bovine strains of the species now occur independently of the eland in association with R. appendiculatus. Its distribution is known to extend at least as far north as Kenya, while at the other end of its range it has been identified in South Africa.

Theileria velifera has the same distribution as T. mutans, including the Caribbean island of Guadeloupe, and is transmitted by at least five African species of Amblyomma ticks, including A. variegatum.

In summary: Africa has all six species, Europe and Asia have T. annulata and T. orientalis, and Australia and the American mainland have T. orientalis, while the Caribbean has been invaded by T. mutans and T. velifera.

Pathogenic Significance

T. parva and T. annulata are among the most virulent pathogens of cattle, while at the opposite extreme, T. velifera infection is asymptomatic. The other species: T. mutans, T. orientalis and T. taurotragi, although usually benign, have some pathogenic potential. The case mortality rate in ECF in European cattle may be over 90 percent. African zebu cattle populations are highly resistant to ECF, provided they were born and bred for generations in endemic areas where all animals acquire infection during calfhood. T. annulata is also very pathogenic for European cattle, with case mortality in dairy cattle as high as 70 percent. Indigenous zebu and taurine cattle populations incur hardly any losses in situations of endemic stability.

T. orientalis appears to be nonpathogenic in most areas, but in eastern Asia and southeastern Asia it may be more pathogenic and has been known to cause some mortality and chronic anemia in European cattle. This higher virulence appears to be strain-associated and may be associated with a rapid rate of division.

In Ethiopia and Burundi, T. orientalis was also associated with disease, but the causal link was not established conclusively.

Theileria mutans probably does little harm, even in European cattle, where there is endemic stability; but serious anemia and even fatal infections have been seen in adult animals exposed for the first time. There are some indications that strains from wild buffalo are more pathogenic than those adapted to cattle.

Theileria taurotragi, long confused with T. mutans and mild T. parva, usually causes no significant disease; but in some animals alarming ECF-like signs may appear, usually followed by recovery. Theileria orientalis, T. mutans, and T. taurotragi may also be aggravating factors in other disease conditions.

As noted above, T. velifera has never been associated with disease.

Signs and Pathogenesis

Disease signs and pathogenesis vary according to the species involved. In ECF, schizonts in lymphocytes become very numerous in acute cases, causing a generalized leucosislike disease and dissemination of infected lymphocytes, leading to generalized hyperplasia of lymph nodes and other lymphoid tissues. Severe leucopenia occurs in the terminal stages of T. parva infection. There is severe immunosuppression, and other infections often complicate the picture. On the other hand, anemia and icterus are the prominent features of acute infections by species with a high rate of multiplication of the erythrocytic piroplasm stage, such as T. mutans and T. orientalis. Hemoglobinuria is not a usual feature. Both the schizont and the piroplasm stages play important roles in the pathogenesis of T. annulata infection. The pathogenesis of Corridor disease, usually fatal in susceptible cattle but associated with very few schizonts and piroplasms, is not understood.

The incubation period of theileriosis varies from less than 10 to over 15 days. Fever is associated with the schizontic phase, limited to a few days with most Theileria, but commonly

persisting until death with T. parva and T. annulata infections. The regional lymph nodes draining the attachment sites of infected ticks swell and this change is followed by swelling of all superficial lymph glands, especially in the case of T. annulata and T. parva. In ECF and Mediterranean theileriosis, symptoms of acute septicemia develop, and breathing becomes difficult in the terminal stage. Death is often due directly to asphyxia following edema of the lungs. Clinical cases of T. orientalis and T. mutans infection, after a short febrile period, show mainly anemia and icterus. A fatal cerebral form of theileriosis, with nervous symptoms, is known as "turning sickness;" it may be caused by T. parva, T. annulata, or T. taurotragi.

Lesions

Depending on the duration of the disease before death, gross lesions in T. parva and T. annulata infections include hyperplasia of lymphoid tissues; edema of the lungs, often with large amounts of froth in the respiratory passages; and characteristic foci of infected lymphoid tissue in the renal cortex, and in cellular walls around ulcers in the abomasal mucosa. Carcasses of animals which have died of Mediterranean theileriosis are usually anemic and icteric. Anemia and icterus are the dominant changes in fatal T. mutans or T. orientalis infections.

Diagnosis

Suspicion of theileriosis on clinical grounds always needs corroboration by microscopic demonstration of the parasites in Giemsa-stained smears of lymph node biopsy material or blood, or both. It should be remembered that a few piroplasms may also be found in the blood of recovered, healthy carriers. Past infection may be demonstrated by serological methods.

Prevention and Control

Tick control by the application of pesticides needs to be done once or twice a week to prevent disease transmission. This makes control very costly. Pesticide application is the main method of ECF control in susceptible breeds of cattle imported into affected countries. Reliable immunization of cattle would make it possible to use less intensive, less costly methods of tick control and would also constitute a last-line defense against a breakdown of chemical tick control due to acaricide resistance, mechanical failure, or human factors.

Immunization with attenuated, cultured schizonts is presently carried out fairly successfully in several countries against T. annulata, but has so far not been effective against T. parva. It is also possible to immunize cattle by using an infection and treatment method: injecting virulent sporozoites from ticks into the cattle and then treating them with tetracyclines at the beginning of the incubation period, or with parvaquone or halofuginone at a later stage.

The existence of important antigenic differences between various strains of T. parva is one factor which has so far prevented its use as an immunizing agent on any significant scale.

Certain antimalarial drugs are curative in theileriosis. Pamaquine and primaquine have been found useful in treating

oriental theileriosis. Other drugs derived from antimalarials have recently been marketed for use against ECF and Mediterranean theileriosis: parvaquone (trade name Clexon) and halofuginone lactate (trade name Lerioxine). Both drugs are usually effective if administered early in the course of disease. Quarantine and slaughter of infected herds has been successful in eradicating ECF from southern Africa, but the problems associated with this approach are usually still insoluble (Dr. G. Uilenberg, University of Utrecht, the Netherlands).

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